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Epidemiological, clinical, and electrophysiological findings in dogs and cats with traumatic brachial plexus injury: A retrospective study of 226 cases

Thibaut Troupel¹ | Nicolas Van Caenegem¹ | Aurélien Jeandel² | Jean-Laurent Thibaud³ | Audrey Nicolle⁴ | Stéphane Blot¹

¹Ecole nationale vétérinaire d'Alfort, Univ Paris Est Créteil, INSERM, IMRB, Maisons-Alfort, France

²Centre Hospitalier Vétérinaire Pommery, Reims, France

³Centre Micen Vet, Créteil, France

⁴Travelling consultant, Mirepoix, France

Correspondence

Stéphane Blot, Ecole Nationale Vétérinaire d'Alfort (EnvA), Unité de Neurologie, 7 avenue du Général de Gaulle, 94700 Maisons-Alfort, France.

Email: stephane.blot@vet-alfort.fr

Abstract

Background: The imaging and electrodiagnostic (EDX) characteristics of traumatic brachial plexus injury (TBPI) are incompletely reported.

Objectives: To describe the epidemiological, clinical, and EDX characteristics of TBPIs in a series of cases in dogs and cats; to determine the association between clinical data, EDX findings, and clinical outcomes; and to assess the sensitivity and specificity of EDX studies to classify nerve lesions.

Animals: One hundred and seventy-five dogs and 51 cats with TBPI and EDX exploration of radial nerve, ulnar nerve, or both nerves.

Methods: Retrospective case series. All medical records were searched for dogs and cats presenting with TBPIs that underwent EDX exploration. Epidemiological, clinical, EDX, and follow-up data were extracted. Association between clinical data, EDX findings, and clinical outcomes was explored.

Results: Forty-six percent of affected animals were injured before 2 years of age and 57% of dogs weighed more than 20 kg. The radial compound muscle action potential (CMAP) amplitude for dogs and cats that had clinical improvement was higher than in animals without improvement (4.3 mV [0-23.6] vs 0 mV [0-2.4], respectively, P = .02). A discriminating radial CMAP amplitude threshold value of 5 mV had a specificity of 93% (95% CI [80-100]) to predict recovery.

Conclusions and Clinical Importance: Electrodiagnostic studies, particularly measurement of radial CMAP amplitude, are valuable diagnostic tests to refine the prognosis of these animals.

KEYWORDS

axonotmesis, electromyography, electroneurography, nerve root avulsion, neurapraxia, neurotmesis, peripheral nerve injury

Abbreviations: BW, body weight; CMAP, compound muscle action potential; EDX, electrodiagnostic; EMG, electromyography; IQR, interquartile range; ROC, receiver operating characteristic; SNEP, sensory nerve evoked potential; TBPI, traumatic brachial plexus injury.

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Diseases affecting the brachial plexus mainly involve inflammatory, neoplastic, or traumatic lesions, with traumatic lesions the most frequent.^{1,2} The clinical diagnosis of traumatic brachial plexus injury (TBPI) is usually easy based on history, clinical signs, and results of neurological examination.³ However, it is challenging to define the prognosis and to determine if the animal will spontaneously recover or if reconstructive surgery or amputation is needed.

Nerve regeneration depends on the type of injury.⁴ The main classification of traumatic nerve injury defines 3 broad categories: neurapraxia, axonotmesis, and neurotmesis,⁵ Neurapraxia is the mildest injury type and includes transient functional loss (conduction block) without loss of nerve continuity. It is associated with an excellent prognosis in days to weeks.⁴ Axonotmesis is defined by the interruption of nerve axons with some degree of myelin loss. The surrounding perineurium and epineurium are preserved.⁴ Axons and myelin located distally to the lesion undergo Wallerian degeneration, but the uninjured shells provide a path for sprouting axons to reinnervate their muscle.⁴ In addition, endoneurium contributes also in promoting axonal regrowth and limiting axonal misrouting.⁴ The prognosis could depend on the degree of axonal loss and the distance between the lesion and target organs because the rate of axonal regeneration is slow, generally estimated at approximately 1 mm per day.⁴ Neurotmesis involves the complete rupture of a nerve (axon, myelin, and surrounding structures). Recovery is not possible without surgical intervention.⁴ From a classification point of view, these definitions apply only to traumatic nerve lesions and do not cover nerve root lesions. Nerve roots have likely a different susceptibility and different responses to traumatic injuries from those of nerves. However, proximal damage associated with axonal loss will lead to a distal axonal degeneration and therefore, nerve changes similar to axonotmesis or neurotmesis. A unique type of nerve root lesion is the nerve root avulsion. It is defined as neurotmesis with axon and myelin sheath rupture occurring at the junction between the spinal cord and both ventral and dorsal nerve roots.⁶ Nerve root avulsion is an irreversible lesion. Therefore, spontaneous improvement can only be observed in animals that have neurapraxia, axonotmesis, or both, in the absence of neurotmesis or nerve root avulsion. Electrodiagnostic (EDX) testing is valuable for the diagnosis of TBPI because it allows more precise identification of the involved nerves than clinical examinations and provides insight into the damage severity.² However, the accuracy of EDX testing for predicting recovery has not yet been determined.

The first aim of this retrospective study was to describe the epidemiological, clinical, and EDX characteristics of TBPI in a case series of dogs and cats. The second aim was to determine the association between clinical data, EDX findings, and clinical outcomes. Another relevant aim was to assess the sensitivity and specificity of EDX studies to classify nerve lesions.

2 MATERIALS AND METHODS

2.1 **Case selection**

The medical records of dogs and cats with history, clinical, and neurological signs consistent with TBPI that underwent EDX exploration with electromyography (EMG) and nerve conduction studies of radial nerve, ulnar nerve, or both nerves presented at the Ecole nationale vétérinaire d'Alfort between 1986 and 2019 were collected. The exclusion criteria were bilateral TBPIs. Epidemiological, clinical, EDX, and follow-up data were reviewed using clinical software (CLOVIS, 4Dv13) and included species, sex, weight, age at injury, age at the time of EDX testing, affected limb, voluntary movements for each joint, Horner's syndrome, cutaneous trunci reflex, sensory testing of autonomous zones, EMG, amplitude of compound muscle action potential (CMAP) and sensory nerve evoked potential (SNEP) of the radial and ulnar nerves, time elapsed between the original trauma and EDX study, and clinical evolution. Surgical findings concerning nerve root avulsion were recorded when available.

2.2 EDX study

All EDX studies were performed at least 7 days after trauma, under general anesthesia by a board-certified neurologist or a neurology resident in training supervised by a board-certified neurologist using similar devices (Nicolet Viking Select & Nicolet Viking IV P, Viasys Healthcare, Madison, Wisconsin).

2.2.1 EMG recording

A disposable bipolar concentric needle electrode was used for EMG analysis (needle 40 mm length, 0.45 mm width, and a 0.068 mm² sampling area). The palmar interosseous, extensor carpi radialis, flexor carpi ulnaris, triceps brachii, biceps brachii, supraspinatus, infraspinatus, and C6 to T2 paraspinal muscles of both the affected and contralateral forelimb were examined.

Amplitude of CMAP and SNEP recording 2.2.2

Polytetrafluoroethylene-coated stainless-steel monopolar electrodes of different lengths with 3 mm bare tips were used for stimulation and recording. Compound muscle action potential was obtained with supramaximal stimuli of 0.1 ms duration, delivered at a frequency of 1 Hz. The CMAP amplitude was recorded from the largest negative peak to the largest positive peak. The CMAP was recorded from the palmar interosseous muscles after stimulation of the distal ulnar nerve and from the extensor carpi radialis muscle after stimulation of the distal radial nerve, as previously described.⁷ For SNEP recording, electrical stimulation was applied as a rectangular wave of 0.1 ms duration at a frequency of 5 Hz with the maximal possible intensity without

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motor interference. At least 100 consecutive recordings were averaged for interpretation. The SNEP amplitude was recorded from the largest negative peak to the largest positive peak. The ulnar SNEP was recorded from the proximal ulnar nerve after subcutaneous stimulation of the lateral part of the fifth digit. The radial SNEP was recorded from the proximal radial nerve after subcutaneous stimulation of the dorsal part of the paw. Neurapraxia was defined by the absence of abnormal spontaneous EMG activity and a normal CMAP amplitude despite obvious clinical nerve dysfunction. Axonotmesis was defined by the presence of abnormal spontaneous EMG activity and decreased CMAP amplitude. Neurotmesis was defined by the presence of abnormal spontaneous EMG activity with the absence of CMAP and SNEP. Nerve root avulsion of the nerve was defined by the presence of abnormal spontaneous EMG activity and the presence of SNEP in the absence of CMAP.

2.3 | Clinical outcomes

Follow-up information was collected during follow-up examinations at the Ecole nationale vétérinaire d'Alfort or by contacting the owners. Animals were included in the improvement group if they can support the weight on the affected limb, ranging from a persistent weightbearing lameness to normal and in the no improvement group if there was no improvement in limb function or if euthanasia or amputation was performed as a direct result of the brachial plexus trauma. The exclusion criteria were animals that had no follow-up information, animals that underwent cross-neurotization, animals that underwent an EDX study more than 3 months after the trauma, and animals classified as "no improvement" with a time between trauma and last follow-up of less than 3 months. The cut-off of 3 months was chosen based on experimental studies on peripheral nerve injuries showing that main axonal regrowth occurs during the first 3 months.⁸⁻¹⁰ The detailed numbers of excluded animals are shown in Figure 1.

2.4 | Statistical analysis

Frequencies and descriptive analyses were performed for species, age at the time of trauma, weight, side of the lesion, neurological deficits, EDX findings, and outcome. For statistical analysis, weight was considered as a binary variable (less or more than 10 kg). Because all variables were non normally distributed, the data are presented as medians and interquartile ranges (IQRs), and nonparametric tests were used for comparisons.

Concerning the relationships between clinical examination and EDX findings, dogs and cats were included in the same statistical analysis. Even if there are species differences in the normal EDX values between these 2 species, when dealing with pronounced nerve lesions, such as frequently observed in TBPIs, CMAP amplitudes are usually so reduced that these species differences are not clinically meaningful. Mann-Whitney and Spearman correlation tests were used to compare epidemiological and clinical data with CMAP amplitude for both the radial and ulnar nerves for qualitative (species, weight,



FIGURE 1 Flow chart showing the composition of the animal groups at each step of the study. Dotted and solid boxes refer to excluded and included animals, respectively

presence of cutaneous trunci reflex presence of Horner's syndrome, and abnormal spontaneous EMG activity in the paraspinal muscles) and quantitative (age and time between trauma and EDX study) data, respectively. Holm-Bonferroni correction was used for univariable analysis and allowed the calculation of an adjusted P(P').

Concerning the relationship between EDX findings and clinical outcomes, dogs and cats were included in the same statistical analysis. Mann-Whitney tests were used to compare CMAP amplitudes for both the radial and ulnar nerves and outcomes. The better discriminating threshold value and the most clinically relevant threshold value of CMAP amplitude were determined when possible. Sensitivity and specificity were calculated for the prediction of further improvement and expressed as percentages and 95% confidence intervals (95% Cls). More precisely, given the previously determined threshold value of CMAP amplitude, the reliability of a greater CMAP amplitude to predict recovery was assessed. Receiver operating characteristic (ROC) curves were calculated when relevant.

Statistical analysis was performed using R software (version 3.6.3, R Foundation for Statistical Computing, Vienna, Austria). For all analyses, P or P' < .05 were considered significant.

3 | RESULTS

3.1 | Descriptive study

3.1.1 | Animals and clinical findings

A total of 226 animals met the inclusion criteria, including 175 dogs (77%) and 51 cats (23%). The group consisted of 105 male (60%) and

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70 female (40%) dogs and 26 male (51%) and 25 female (49%) cats. The dog group comprised many breeds, with crossbreeds (44/175), German shepherds (18/175), and Labrador retrievers (15/175) the most common. Body weight (BW) was available for 168 dogs; of these, 8 weighed less than 5 kg (5%), 21 weighed 5 to 10 kg (13%), 40 weighed 10 to 20 kg (24%), 76 weighed 20 to 30 kg (45%), and 23 weighed more than 30 kg (14%). The cat group comprised only 2 purebreds (1 British shorthair and 1 Siamese); the other cats were European shorthairs. Body weight was available for 51 cats; of these, 48 had a BW of 5 kg or less (94%) and 3 had a BW of 5 to 10 kg (6%).

The age at the time of trauma was available in 171 dogs and 50 cats, with a median of 26 months (IQR [12-63]) for dogs and 24 months (IQR [9-48]) for cats. The age distribution is shown in Figure 2. Forty-six percent of the animals were injured before 2 years of age. Data on the side of the lesion were available for 175 dogs, with 104 (59%) and 71 dogs (41%) having left- and right-sided lesions, respectively. Data on the side of the lesion were available for all cats, with 26 (51%) and 25 cats (49%) having left- and right-sided lesions, respectively.

Sensory testing of autonomous zones of radial, ulnar, median, and musculocutaneous nerves were available for 166 dogs and 49 cats. One hundred and fifty dogs (90%) and 41 cats (84%) had anesthesia of the radial autonomous zone. One hundred and forty-seven dogs (89%) and 42 cats (86%) had anesthesia of the ulnar autonomous zone. One hundred and eleven dogs (67%) and 33 cats (67%) had anesthesia of the median autonomous zone. Sixty-eight dogs (41%) and 15 cats (31%) had anesthesia of the musculocutaneous autonomous zone. Sixty-six dogs (40%) and 13 cats (27%) had anesthesia of all the previously named autonomous zones.

The description of limb movements was available for 133 dogs and 46 cats. Fifty dogs (38%) and 12 cats (26%) had monoplegia, defined by paralysis of shoulder, elbow and carpus. Animals with voluntary movements in 1 or more joints were classified as having monoparesis. It concerned 83 dogs (62%) and 34 cats (74%). In details, in dogs, 87 (65%) and 90 (68%) did not have flexion or extension of the shoulder, respectively and 79 dogs (59%) had shoulder paralysis. Seventy-two (54%) and 113 dogs (85%) did not have flexion or extension of the elbow, respectively and 69 dogs (52%) had elbow paralysis. One hundred and twenty-six (95%) and 117 dogs (88%) did not have flexion or extension of the carpus, respectively and 114 dogs (86%) had carpus paralysis. In cats, 24 (52%) and 26 (57%) did not have flexion or extension of the shoulder, respectively and 21 cats (46%) had shoulder paralysis. Nineteen (41%) and 37 cats (80%) did not have flexion or extension of the elbow, respectively and 17 cats (37%) had elbow paralysis. Forty-five (98%) and 43 cats (93%) did not have flexion or extension of the carpus, respectively and 43 cats (93%) had carpus paralysis.

Data concerning Horner's syndrome and the presence of cutaneous trunci reflex were available for 161 dogs and 50 cats and 160 dogs and 46 cats, respectively. Sixty-eight dogs (42%) and 19 cats (38%) had Horner's syndrome on the eye on the same side as the lameness. All the affected animals had anisocoria with myosis on the side of the lameness, whereas other clinical signs of Horner's syndrome were inconsistent. One hundred and thirty dogs (81%) and 38 cats (83%) had lost the cutaneous trunci reflex on the same side as

3.1.2 | EDX testing

the lameness.

Complete EMG data of the affected limb were available in 147 dogs and 46 cats. All animals showed abnormal spontaneous EMG activity in at least 1 muscle. Table 1 shows the distribution of abnormal spontaneous EMG activity for both dogs and cats.

Compound muscle action potential and SNEP recordings of the radial and ulnar nerves were available for 158 dogs and 46 cats and 151 dogs and 46 cats, respectively. For dogs, the median CMAP amplitude of the radial and ulnar nerves was 0 mV (IQR [0-10]) and 0 mV (IQR [0-3]), respectively. For cats, the median CMAP amplitude of the radial and ulnar nerves was 1 mV (IQR [0-12]) and 0 mV (IQR [0-0]), respectively. Forty-three dogs (27%) and 13 cats (28%) had EDX features of nerve root avulsion of the radial nerve and 54 dogs (34%) and 9 cats (20%) had radial neurotmesis. Forty-one dogs (27%) and 21 cats (46%) had EDX features of nerve root avulsion of the ulnar nerve and 59 dogs (39%) and 19 cats (41%) had ulnar neurotmesis.

3.1.3 | Surgical findings

Data concerning macroscopic nerve lesions during cross-neurotization surgery or amputation were available for 15 animals. All cases were classified as neurotmesis or nerve root avulsion based on EDX findings. Nine animals with suspected nerve root avulsion were confirmed in surgery, whereas 6 animals with EDX findings consistent with neurotmesis were found to have nerve root avulsion. These results suggested that while EDX testing is highly specific for the diagnosis of nerve root avulsion, it lacked sensitivity, leading to misdiagnosis of neurotmesis. No case was documented with surgical findings consistent with partial nerve lesions such as axonotmesis.

3.2 | Statistical analysis

3.2.1 | Correlation between clinical and EDX findings

After excluding animals with incomplete data for any study data, this analysis included 129 dogs and 42 cats.

For the radial nerve, ipsilateral absence of cutaneous trunci reflex and abnormal spontaneous EMG activity in the paraspinal muscles were associated with decreased CMAP amplitude (Table 2). Concerning quantitative data, we observed a positive correlation between the time between trauma and EDX examination and CMAP amplitude of the radial nerve (r = .31, P' = .001). When the time between trauma and EDX examination increased, the amplitude of radial CMAP also increased.

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FIGURE 2 Animal ages at the time of plexus brachial traumatic injury onset

 TABLE 1
 Distribution of abnormal electromyographic activity on the affected limb

Muscle	D	%	с	%
Palmar interosseous	133	90	44	96
Extensor carpi radialis	136	93	43	93
Flexor carpi ulnaris	132	90	42	91
Triceps brachii	132	90	40	87
Biceps brachii	96	65	24	52
Infraspinatus	87	59	21	46
Supraspinatus	86	59	18	39
C6 to T2 paraspinal	88	60	23	50

Abbreviations: *C*, number of cats with abnormal electromyographic activity in each muscle; *D*, number of dogs with abnormal electromyographic activity in each muscle.

For the ulnar nerve, species, weight, and ipsilateral absence of cutaneous trunci reflex, but not CMAP, were associated with decreased CMAP amplitude (Table 2).

Multivariable regressions were attempted using linear and logistic regressions after data transformation; however, no model reached assumptions after analysis of the residuals.

3.2.2 | Prognostic factors

The group contained 30 animals (15 dogs and 15 cats) and all of them presented a nonweight-bearing lameness at the time of EDX study. Fifteen animals showed improvement and 15 showed no improvement. The retrospective nature of this study did not allow an exploration of the associations between clinical examination findings and clinical outcomes but we explored the correlation between EDX findings and clinical outcomes.

The radial nerve CMAP amplitude of the animal with improvement was significantly higher than that of animals without improvement (4.3 mV, IQR [0-23.6] vs 0 mV, IQR [0-2.4], P = .02). In contrast, the CMAP amplitude of the ulnar nerve did not differ between animals with and without improvement (0 mV, IQR [0-1.3] vs 0 mV, IQR [0-0], P = .16). The calculated discriminating threshold CMAP amplitude value of the radial nerve was 0.7 mV which showed a sensitivity of 80% (95% CI [60-100]) and a specificity of 67% (95% CI [40-87]). The area under the ROC curve was 0.73 (95% CI [55-90]; Figure 3). A threshold radial CMAP amplitude value of 5 mV also provided valuable clinical results (Figure 4), with a sensitivity of 47% (95% CI [20-73]) and a specificity of 93% (95% CI [80-100]) to predict further improvement. Considering the high proportion of excluded animals which is mainly because of the lack of follow-up, the authors highlight that the respective proportion of improvement or no improvement was probably altered by the selection criteria. A specificity of 93% (95% CI [80-100]) is sufficiently high to consider a high probability for further improvement in animals with CMAP amplitude greater than 5 mV.

4 | DISCUSSION

Determining the prognosis of animals with both motor and sensory traumatic dysfunction of 1 or more nerves is challenging because it 2842 Journal of Veterinary Internal Medicine



Nerves	Variables		CMAP amplitude (median (mV) [IQR])	P'
Radial	Specie	Dog	0 [0-10.7]	1
		Cat	0.05 [0-5.2]	
	Weight	Less than 10 kg	0 [0-6.2]	1
		More than 10 kg	0 [0-10.6]	
	CTR	Present	11.5 [1.7-25]	<.001*
		Absent	0 [0-3]	
	HS	Present	0 [0-2.1]	.06
		Absent	1 [0-14.3]	
	PS	Present	0 [0-2.9]	.04*
		Absent	1.5 [0-14.1]	
Ulnar	Specie	Dog	0 [0-4]	.05*
		Cat	0 [0-0]	
	Weight	Less than 10 kg	0 [0-0.13]	.05
		More than 10 kg	0 [0-5]	
	CTR	Present	4.4 [0-15.9]	.001*
		Absent	0 [0-1.8]	
	HS	Present	0 [0-1.2]	.26
		Absent	0 [0-5.4]	
	PS	Present	0 [0-2.2]	.67
		Absent	0 [0-4.5]	

TABLE 2 Univariable analysis of clinical and electrodiagnostic findings in function of radial and ulnar compound muscle action potential (CMAP) amplitude

Note: Values of P' < .05 are indicated by *.

Abbreviations: CTR, cutaneous trunci reflex; HS, Horner's syndrome; PS, abnormal spontaneous

electromyographic activity in the paraspinal muscles.



FIGURE 3 Receiver operating characteristic (ROC) curve of radial compound muscle action potential (CMAP) amplitude for animals without improvement. The gray area represents the 95% confidence interval. The solid gray line represents the null hypothesis (area under the ROC curve = 0.5)

depends on the severity of nerve injury with 3 possibilities: neurapraxia, axonotmesis, and neurotmesis.⁵ Clinically, it is almost impossible to distinguish between these 3 categories unless recovery occurs, but it can take many months if occurring at all. Thus, it is of



FIGURE 4 Distribution of radial compound muscle action potential (CMAP) amplitudes for animals with and without clinical improvement

the utmost importance to perform ancillary testing, such as EDX examination, to determine the degree of neural loss to accurately suggest surgical intervention for neural repair or amputation.^{2,3,6,11-15} In our study, the comparison of EDX and surgical findings showed that some animals with nerve root avulsion could be misdiagnosed with neurotmesis because SNEP was not elicited during the examination.

However, every animal with an EDX diagnosis of nerve root avulsion was confirmed by surgical findings. Sensory nerve evoked potential recording is challenging in the presence of abnormal spontaneous EMG activity from neighboring denervated muscles. The abnormal spontaneous EMG activity can completely obscure the SNEP as its amplitude (10-4000 μ V when the recording is performed within the muscle) is greater than the 1 of the SNEP (usually around 10 μ V). Moreover, trauma can lead to multiple lesions within the same nerve with combination of nerve root avulsion and distal neurotmesis or combination of nerve root avulsion and conduction block in the distal process of the sensory neurons or their degeneration. These lesions lead to an absence of SNEP recording during EDX study despite the presence of nerve root avulsion and can explain the high specificity but the lack of sensitivity of SNEP for the diagnosis of nerve root avulsion. Considering the poor prognosis of both neurotmesis and nerve root avulsion, these misdiagnoses should not have a clinical impact unless surgical treatment is considered.

In peripheral nerve lesions, the presence of abnormal spontaneous EMG activity allows the exclusion of neurapraxia. However, for a damaged nerve examination, the absence of abnormal spontaneous EMG activity can either reflect neurapraxia or severe muscle atrophy or fibrosis because of denervation. The distinction between these 2 opposite situations is based on clinical examination and CMAP assessment. The CMAP amplitude directly reflects the number of responding muscle fibers innervated by the tested nerve.^{7,13} Hence. in case of a reduced CMAP amplitude with abnormal spontaneous EMG activity, the reduction of CMAP amplitude could highlight the degree of axonal loss after trauma. According to CMAP amplitude, we can classify lesions as minor axonotmesis with normal/subnormal CMAP amplitude or severe axonotmesis with severely decreased CMAP amplitude or neurotmesis with no elicited CMAP.^{7,13} The use of monopolar needle recording electrodes can sometimes allow the detection of a distant signal from muscle innervated by nonaffected nerves and the identification of a far-field potential that can be confounded with CMAP (S. Blot, personal observation). To minimize such errors, we recommend moving the recording needle electrodes slightly after CMAP recording. If CMAP remains strictly the same despite this movement, distant signal should be considered. Another method is to repeat recording with a bipolar concentric needle electrode to register only a small muscle sample. In our study, animals with CMAP recording consistent with a far-field potential were considered as they had no CMAP.

Concerning the prediction of nerve damage based on clinical data, we showed that the absence of ipsilateral cutaneous trunci reflex was associated with a significantly lower CMAP amplitude of both the radial and ulnar nerves and, thus, with a more important axonal lesion for these nerves. The preganglionic neurons of the sympathetic innervation of the eye leave the spinal cord together with the C8 to T4 ventral nerve roots. As they emerge from the intervertebral foramen, they leave the spinal nerve in the segmental ramus communicans, which joins the thoracic sympathetic trunk. Besides, the radial nerve is formed by C7 to T2 nerve roots in most dogs. Therefore, both Horner's syndrome and radial nerve injury are likely associated. Of merican College of

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note, a Horner's syndrome supports that lesions exist within the spinal canal.¹⁶ We also showed that some other clinical variables can interfere with these data, such as the time elapsed between trauma and EDX study. The increase of the CMAP amplitude of radial nerve with time might reflect either an improvement of the injured innervation (resolution of conduction block or axonal regrowth) or collateral reinnervation from remaining intact axons within their muscle fascicles that occurs within days to few weeks after trauma. Therefore, an increase in CMAP amplitude with time does not necessarily reflects a functional improvement. One can relevantly consider that the animal's weight at the time of trauma is an aggravating factor for nerve lesions because, the heavier the animal is, the stronger the inertia of the body might increase the nerve traction during trauma. Our study did not find such a correlation between weight and pronounced lesions of the nerves. The distance between the nerve lesion and the target muscle might be a prognostic factor as the time needed for axonal sprouting and thus, reinnervation depends on this distance but this would require a prospective study taking into account these data. Moreover, experimental and human studies showed that the proportion of regenerating axons progressively decreased with time.8-10 In view of using this variable, the distance between the nerve lesion and the target muscle could be approximated using the distance between standardized conventional stimulation points during EDX study. Neuropathic paresthesia or pain has been occasionally associated with TBPI but these data had not been recorded. However, such information could be relevant in further studies. The presence of abnormal spontaneous EMG activity in the paraspinal muscles was associated with a more severe lesion of the radial nerve. Interestingly, there is an association between abnormal spontaneous EMG activity in the paraspinal muscles and nerve root avulsion of the ulnar nerve.⁶ Our study showed that radial CMAP amplitude was predictive of recovery despite moderate sensitivity and specificity. The ulnar CMAP amplitude was less informative, which is meaningful because the radial nerve is of utmost importance for weight-bearing in guadrupedal animals.¹ We also determined an optimal radial CMAP amplitude cut-off to predict a lack of improvement. While a radial CMAP amplitude of less than 0.7 mV has a sensitivity of 80% (95% CI [60-100]) to predict no further improvement, the use of a different threshold value also leads to clinically valuable results: a radial CMAP amplitude threshold of 5 mV showed a good specificity of 93% (95% CI [80-100]), meaning that a large proportion of animals with a radial CMAP amplitude above 5 mV will improve with time.

A limitation of our study was that the recording of radial CMAP was only performed in the extensor carpi radialis muscle and not in the triceps brachii. It seems reasonable to consider that a dog could display a severe lesion (such as neurotmesis) of the radial nerve branch that innervates the extensor carpi radialis muscle and only a partial (such as axonotmesis) lesion of the radial nerve branch innervating the triceps brachii. In such cases, EDX testing could indicate neurotmesis but the animal could recover triceps brachii function. Thus, the evaluation of triceps brachii response or the identification of far-field potential originating from this muscle and recorded in the extensor carpi radialis muscle could be interesting in animals in



which no CMAP can be elicited in the extensor carpi radialis muscle.

Magnetic stimulation of the radial nerve in dogs with TBPI is a good prognostic indicator for recovery and can be performed earlier than EMG.¹² Practically, it is important to wait several days after the injury before performing EMG recording to allow the denervated myofibers to become hypersensitive and produce abnormal electrical activity.^{7,17} An experimental study in dogs showed that the optimal delay after nerve injury to record maximal changes during EMG was 8 to 10 days.^{17,18} While magnetic stimulation can be performed earlier, this is also true for studies of nerve conduction velocity, which is affected as soon as the injury occurs.⁷ To date, there is no evidence that magnetic stimulation or EDX examination is better to predict recovery. A similar report evaluating magnetic stimulation is needed to compare these methods and to determine whether the combination of both methods is better than either alone. While some imaging methods in human medicine could diagnose nerve root avulsions in TBPI, such as computed tomography myelography and diffusion tensor imaging,^{19,20} to date, no similar reports exist in veterinary medicine.

Most of the limitations of this study relate to its retrospective nature, leading to the exclusion of numerous animals because of incomplete clinical data or loss to follow-up. As we observed that the time between trauma and EDX study affected the CMAP recording, we censored animals that were tested more than 3 months after the trauma. While this likely increased the accuracy of our results, an important number of animals were excluded from the prognostic factor study. In our study that explore the clinical and EDX features of TBPIs in both dogs and cats, the results showed that EDX testing is a valuable tool to refine the prognosis of these lesions and can predict the evolution of nerve lesions.

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CONFLICT OF INTEREST DECLARATION

Authors declare no conflict of interest.

OFF-LABEL ANTIMICROBIAL DECLARATION

Authors declare no off-label use of antimicrobials.

INSTITUTIONAL ANIMAL CARE AND USE COMMITTEE (IACUC) OR OTHER APPROVAL DECLARATION

Authors declare no IACUC or other approval was needed.

HUMAN ETHICS APPROVAL DECLARATION

Authors declare human ethics approval was not needed for this study.

ORCID

Thibaut Troupel D https://orcid.org/0000-0001-8844-2214 Nicolas Van Caenegem 🕩 https://orcid.org/0000-0003-0185-7738 Aurélien Jeandel D https://orcid.org/0000-0002-8097-4088 Stéphane Blot D https://orcid.org/0000-0002-8982-0317

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